11:45

IMPAIRED UTILIZATION OF CORONARY VASCULAR RESERVE IN HYPERCHOLESTEROLEMIC SWINE

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In-vitro studies document altered vasoreactivity of coronary resistance vessels exposed to hypercholesterolemia. Accordingly, we evaluated the ability to augment coronary blood flow (CBF) in 17 consecutive swine (sw) [10 normal(NL) and 7 hypercholesterolemic for 1-3 mos (HC)] during periods of increased oxygen demand and after selective thromboxane inhibition with UK38,485 (UK). Regional CBF (ml/min/g) was measured using radioactive microspheres in closed chest sw distal to an 80% LAD stenosis and in the normal circumflex (circ) zone. Cholesterol levels were elevated in HC sw (279±94 vs 124±22, mg/dl, p<.002). Double product was mildly elevated in HC sw at baseline compared to NL sw (12,361±1277 vs 11,172±1112, p=.06) and increased by a similar amount with pacing (23% vs 18%, p<.001 vs B). Results (CBF, mean ± SD):

Zone	Baseline	Paced	Post UK
NL circ	0.88±.18	1.07±.35*	1.02±.19*
LAD	0.84±.21	1.02±.37+	1.01±.28*
HC circ	1.24±.41$	1.02±.37	1.15±.29
LAD	1.07±.37	0.86±.24	0.97±.27

*p<.06, +p=.07 vs Baseline, $p=.025 vs NL

Comparison of the response to pacing between NL and HC sw reveals an appropriate increase in NL sw which was absent in HC sw (.19±.29 vs -0.22±.27 circ, .19±.29 vs -.21±16 LAD, p<.01). A similar response occurred to UK, most apparent in the circ zone (.14±.21 vs -.15±.38, p=.06). These data document impaired utilization of coronary vascular reserve in animals exposed to a relatively brief period of hypercholesterolemia.

10:45

OUTCOME OF PATIENTS WITH REFRACTORY VENTRICULAR TACHYARRHYTHMIAS DECLINING AUTOMATIC IMPLANTABLE CARDIOVERTER-DEFIBRILLATOR

Dirk Behringer, Diana Aarons, Enrico P. Veltri, Sinai Hospital of Baltimore, Baltimore, MD

The automatic implantable cardioverter-defibrillator (AICD) has had a marked impact on the outcome of patients at high risk for sudden cardiac death (SCD). No study to date however has assessed the clinical outcome of patients with refractory ventricular tachycardia (VT)/fibrillation (VF) declining AICD implantation. We followed 18 patients with drug-refractory VT/VF evaluated during March 1983 through July 1990 who were recommended AICD implantation after drug failure by serial programmed electrical stimulation (PES), but who refused surgery. Patient characteristics were: males 16, mean age 69 years (34-87), mean ejection fraction 30%, coronary artery disease 15, valvular disease 2, cardiomyopathy 1. The patients were discharged on "best" antiarrhythmic drug therapy as evaluated by PES where applicable. RESULTS: Fifteen patients had inducible sustained VT off antiarrhythmic drugs and failed a mean 3 drugs, 3 patients had noninducible VT/VF. Eleven patients were discharged on amiodarone, 5 patients on Class I drugs, and 1 on beta-blocker. One patient died of refractory heart failure pre-discharge. At mean 14 months (1-35) followup, 6 (35%) patients discharged were alive, 11 were dead: SCD 7 (41%) (3 on amiodarone), congestive heart failure 2, noncardiac 2. Five patients without SCD had recurrent documented sustained VT/VF with resuscitation (2) or syncope (3). Overall clinical recurrence of VT/VF occurred in 12 patients (71%). We conclude that in patients with drug-refractory VT/VF in whom AICD has been recommended but declined, long term prognosis is poor despite "best" drug therapy.

Thursday, March 7, 1991
10:30AM–12:00NOON, Room 367, West Concourse
Cardiac Defibrillation II

10:30

EMERGENCY INTRACARDIAC DEFIBRILLATION IN PATIENTS WITH REFRACTORY VENTRICULAR FIBRILLATION

Todd J. Cohen, Melvin M. Scheinman, Booker T. Pullen, Nancy A. Chiesa, Michael D. Lesh, John M. Herre, Michael A. Lee, Jerry C. Griffin, University of California, San Francisco, CA

Ventricular fibrillation (VF) refractory to cardiopulmonary resuscitation and multiple transthoracic defibrillations occurred in 3 pts during 2237 consecutive invasive electrophysiological studies (from 1/87 to 9/90). Pts' mean age was 66±7 yrs; and all had severe coronary disease with LV dysfunction (EF=30±3%). All had recurrent monomorphic ventricular tachycardia (VT); 2 had chronic obstructive pulmonary disease and 1 was obese. All were being treated with antiarrhythmic agents (amiodarone; amiodarone and mexiletine; and procainamide and mexiletine) at time of study. In each, stable monomorphic VT (CLs = 320, 350, and 570 ms) was initially induced, and overdrive ventricular pacing accelerated the VT to VF. Multiple transthoracic defibrillations (7 to 15/pt, using up to 500 J) failed to terminate these arrhythmias. As a last resort, intracardiac defibrillation was performed using a standard defibrillator connected to a 6 Fr RV quadripolar catheter (distal pole = cathode), and posterior skin patch (anode). The first pt had prolonged resuscitation (>35 min) prior to application of intracardiac shocks. Two intracardiac shocks (200 and 500 J) converted VF to sinus rhythm (though this pt remained in sinus rhythm, he died an hour later of severe pump failure). Two pts received a single intracardiac defibrillation (100 and 300 J), within 15 min of resuscitation, which immediately converted VF to sinus rhythm. Both of these pts are alive and well with a mean follow up of 11±9 months.

Conclusion: Intracardiac defibrillation proved useful in terminating refractory VF, particularly when applied early (<15 min) in the course of cardiac resuscitation resistant to multiple high energy transthoracic defibrillations.

11:00

IMPLANTABLE DEFIBRILLATORS: ANTIARRHYTHMIC THERAPY OF CHOICE IN HIGH RISK PATIENTS WITH DILATED CARDIOMYOPATHY

Patrick J. Tchou, Anita C. Krebs, Kathi Axtell, Jasbir Sra, Mohammad Jazayeri, Paul Troup, Masood Akhtar, Sinai Samaritan Medical Center, Milwaukee, WI

Antiarrhythmic drug therapy as well as implantable automatic defibrillators (IAD) are modalities of therapy available to treat patients (Pts) with dilated cardiomyopathies (DCM). We analyzed the outcome of 84 DCM Pts who underwent electrophysiology studies (EPS) at our center. Group I included 11 pts treated with implantable automatic defibrillators (IAD). Ten of these 11 presented with sudden death (SCD), sustained VT or syncope with inducible sustained monomorphic VT. Group II presented similarly with SCD, sustained VT, or syncope, but were treated with EPS guided drug therapy. Group III included pts with dizziness or lesser symptoms, and no documented sustained VT. Drug therapy in group III, if any, were also guided by EPS results. Clinical variables and follow-up durations were similar in all groups. Results are as follows:

	Group I (N=11)	Group II (N=39)	Group III (N=34)
sudden death	0(0%)*	9(23%)	1(3%)
Other deaths	1(9%)	7(18%)	8(24%)

* p<0.01 vs groups II

Five (45%) of Group I Pts received appropriate shocks during follow-up.

Conclusions: IAD therapy appears to be superior to EPS guided drug therapy in preventing SCD among DCM Pts who initially present with SCD, sustained VT or syncope.